COPD

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Definition

- Chronic Obstructive Pulmonary Disease (COPD) is a common, preventable and treatable disease that is characterized by persistent respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases.

Prevalence of COPD

- Estimated 384 million COPD cases in 2010.
- Estimated global prevalence of 11.7% (95% CI 8.4%–15.0%).
- Three million deaths annually.
- With increasing prevalence of smoking in developing countries, and aging populations in high-income countries, the prevalence of COPD is expected to rise over the next 30 years.
- By 2030 predicted 4.5 million COPD related deaths annually.

mortality ,Economic and Social Burden

- COPD was the third leading cause of death in the United States.
- Disability-Adjusted Life Year (DALY) = sum of years lost because of premature mortality and years of life lived with disability, adjusted for the severity of disability.
- COPD is an increasing contributor to disability and mortality around the world.
- In 2013 COPD was 5th leading cause of DALYs lost.
- In the United States, COPD is the second leading cause of reduced DALYs, trailing only ischemic heart disease

Risk factors

Environmental

- Tobacco smoke accounts for 95% of cases in UK
- Indoor air pollution; cooking with biomass fuels in confined areas in developing countries
- Occupational exposures, such as coal dust, silica and cadmium
- Low birth weight may reduce maximally attained lung function in young adult life
- Lung growth: childhood infections or maternal smoking may affect growth of lung during childhood, resulting in a lower maximally attained lung function in adult life
- Infections: recurrent infection may accelerate decline in FEV_1; persistence of adenovirus in lung tissue may alter local inflammatory response, predisposing to lung damage; HIV infection is associated with emphysema
- Low socioeconomic status
- Cannabis smoking

Host factors

- Genetic factors: α_1-antiproteinase deficiency; other COPD susceptibility genes are likely to be identified
- Airway hyper-reactivity

Pathology, pathogenesis & pathophysiology

Pathology, pathogenesis & pathophysiology

- **Pathogenesis**
- The inflammation observed in the respiratory tract of COPD patients appears to be a modification of the normal inflammatory response of the respiratory tract to chronic irritants such as cigarette smoke.
  - Oxidative stress Oxidants (hydrogen peroxide, 8-isoprostane) are both generated by cigarette smoke and other inhaled particulates, and released from activated inflammatory cells such as macrophages and neutrophils. There may also be a reduction in endogenous antioxidants in COPD patients. Oxidative stress be an important amplifying mechanism in COPD.

Pathology, pathogenesis & pathophysiology

- Protease-antiprotease imbalance.
- There is compelling evidence for an imbalance in the lungs of COPD patients between proteases(derived from inflammatory cells and epithelial cells) that break down connective tissue components and antiproteases that counterbalance this Action
- Unopposed action of proteases and oxidants leading to destruction of alveoli and appearance of emphysema.
- Increased inflamatory cells such as macrophages , activated neutrophils and increased lymphocytes release multiple inflammatory mediators that amplify the inflammatory process (proinflammatory cytokines), and induce structural changes.

Pathology, pathogenesis & pathophysiology

- Inflammation may precede the development of fibrosis or repeated injury of the airway wall itself may lead to excessive production of muscle and fibrous tissue. This may be a contributing factor to the development of small airways limitation and eventually the obliteration that may precede the development of emphysema.
- Loss of elastic tissue, inflammation and fibrosis in airway wall result in premature airway closure, gas trapping and dynamic hyperinflation leading to changes in pulmonary and chest wall compliance.
- Consequently ,decrease ventilation and Gas exchange abnormalities result in hypoxemia and hypercapnia.
- Inflammatory mediators in the circulation may contribute to skeletal muscle wasting and cachexia, and may initiate or worsen comorbidities.

Clinical features

- **SYMPTOMS**
  - Cough
- Chronic cough is often the first symptom of COPD and is frequently discounted by the patient as an expected consequence of smoking and/or environmental exposures. Initially, the cough may be intermittent, but subsequently may be present every day, often throughout the day. Chronic cough in COPD may be productive or unproductive.
- In some cases, significant airflow limitation may develop without the presence of a cough.
- Dyspnea, a cardinal symptom of COPD, is a major cause of the disability and anxiety that is associated with the disease. Typical COPD patients describe their dyspnea as a sense of increased effort to breathe, chest heaviness,air hunger, gasping

Clinical features

- ***Sputum production.***
- COPD patients commonly raise small quantities of tenacious sputum with coughing. Regular production of sputum for three or more months in two consecutive years (in the absence of any other conditions that may explain it) is the classical definition of chronic bronchitis.
- sputum production can be intermittent with periods of flare-up interspersed with periods of remission. Patients producing large volumes of sputum may have underlying bronchiectasis.

❖ ***Wheezing and chest tightness.***

- Wheezing and chest tightness are symptoms that may vary between days, and over the course of a single day.
- An absence of wheezing or chest tightness does not exclude a diagnosis of COPD.

Clinical features

- Other symptoms
- Haemoptysis may complicate exacerbations of COPD but should not be attributed to COPD without thorough investigation.
- Fatigue, weight loss and anorexia are common problems in patients with severe and very severe COPD. They have prognostic importance and can also be a sign of other diseases, such as tuberculosis or lung cancer, and therefore should always be investigated.
- Ankle swelling in core pulmonale
- Cough syncope ,fracures
- Depression,anxiety are common ,should be always sought.

History

- A detailed medical history of a new patient who is known, or suspected, to have COPD should include:

► Patient's exposure to risk factors, such as smoking and occupational or environmental exposures.

► Past medical history, including asthma, allergy, sinusitis, or nasal polyps; respiratory infections in childhood; other chronic respiratory and non-respiratory diseases.

► Family history of COPD or other chronic respiratory disease.

► Pattern of symptom development: COPD typically develops in adult life and most patients are conscious of increased breathlessness, more frequent or prolonged "winter colds," and some social restriction for a number of years before seeking medical help.

History

► History of exacerbations or previous hospitalizations for respiratory disorder. Patients may be aware of periodic worsening of symptoms even if these episodes have not been identified as exacerbations of COPD.

► Presence of comorbidities, such as heart disease, osteoporosis, musculoskeletal disorders, and malignancies that may also contribute to restriction of activity.

► Impact of disease on patient's life, including limitation of activity, missed work and economic impact, effect on family routines, feelings of depression or anxiety, well-being and sexual activity.

► Social and family support available to the patient.

► Possibilities for reducing risk factors, especially smoking cessation.

Physical examination

- Physical signs are non-specific, correlate poorly with lung function, and are seldom obvious until the disease is advanced.
- General examination
- Cyanosis,plethoric face(polycythemia),cachexia in advance ,flapping tremor,clubbing is not a feature of COPD (needs investigations),ll oedema ,
- Local examination
- Distress,using accesory muscles,increased AP diameter,hyper resonance ,liver dulness is below ,HVB,rhonchi bilateral .
- Increased P2

Chronic bronchitis versus emphesema .

- In practice, these phenotypes often overlap.

Chronic bronchitis versus emphesema

	Chronic bronchitis	emphesema
SYMPTOMS	Predominantly cough	Predominantly SOB
Appearance	Blue bloater'	Pink puffer
Body build	obese	thin
Cyanosis	predominant	Absent early
Cor pulmonale	early	late
Diagnosis	clinical	radiological
radiology	Hyperinflation not common	Hyperinflation common
pco2	Early increased	Early normal
Transfer factor KCO	normal	decreased

Investigations

- Spirometry

- Diagnosis :objective demonstration of airflow obstruction by spirometry and is established when the postbronchodilator FEV1/FVC is < 70%.
- Assesment of severity of obstruction(prognosis)
- Follow up assesment
- Measurement of lung volumes

The presence of emphysema is suggested by a low gas transfer and DLCO .

- Exercise testing and assessment of physical activity.

Assesing response ,prognosis

- Pulse oximetry and ABG
- CBC (polycythemia)

Investigations

- Imaging.
- A chest X-ray is not useful to establish a diagnosis in COPD, but it is valuable in excluding alternative diagnoses and establishing the presence of significant comorbidities such as concomitant respiratory (pulmonary fibrosis, bronchiectasis, pleural diseases), skeletal (e.g., kyphoscoliosis), and cardiac diseases (e.g., cardiomegaly).
- Radiological changes associated with COPD include signs of lung hyperinflation (flattened diaphragm and an increase in the volume of the retrosternal air space), hyperlucency of the lungs, and rapid tapering of the vascular markings.
- Computed tomography (CT) of the chestis not routinely recommended except for detection of bronchiectasis,exlude DDX ,prior to surgery ,transplant .

Investigations

- Alpha-1 antitrypsin deficiency (AATD) screening.
- In young pt less than 45 ys,panacinar basal emphysema .

DIFFERENTIAL DIAGNOSIS OF COPD

DIAGNOSIS	SUGGESTIVE FEATURES
COPD	Onset in mid-life. Symptoms slowly progressive. History of tobacco smoking or exposure to other types of smoke.
Asthma	Onset early in life (often childhood). Symptoms vary widely from day to day. Symptoms worse at night/early morning. Allergy, rhinitis, and/or eczema also present. Family history of asthma. Obesity coexistence.
Congestive Heart Failure	Chest X-ray shows dilated heart, pulmonary edema. Pulmonary function tests indicate volume restriction, not airflow limitation.
Bronchiectasis	Large volumes of purulent sputum. Commonly associated with bacterial infection. Chest X-ray/CT shows bronchial dilation, bronchial wall thickening.
Tuberculosis	Onset all ages. Chest X-ray shows lung infiltrate. Microbiological confirmation. High local prevalence of tuberculosis.
Obliterative Bronchiolitis	Onset at younger age, nonsmokers. May have history of rheumatoid arthritis or acute fume exposure. Seen after lung or bone marrow transplantation. CT on expiration shows hypodense areas.
Diffuse Panbronchiolitis	Predominantly seen in patients of Asian descent. Most patients are male and nonsmokers. Almost all have chronic sinusitis. Chest X-ray & HRCT show diffuse small centrilobular nodular opacities & hyperinflation.

These features tend to be characteristic of the respective diseases, but are not mandatory. For example, a person who has never smoked may develop COPD (especially in the developing world where other risk factors may be more important than cigarette smoking); asthma may develop in adult and even in elderly patients.

OTHER CAUSES OF CHRONIC COUGH

INTRATHORACIC

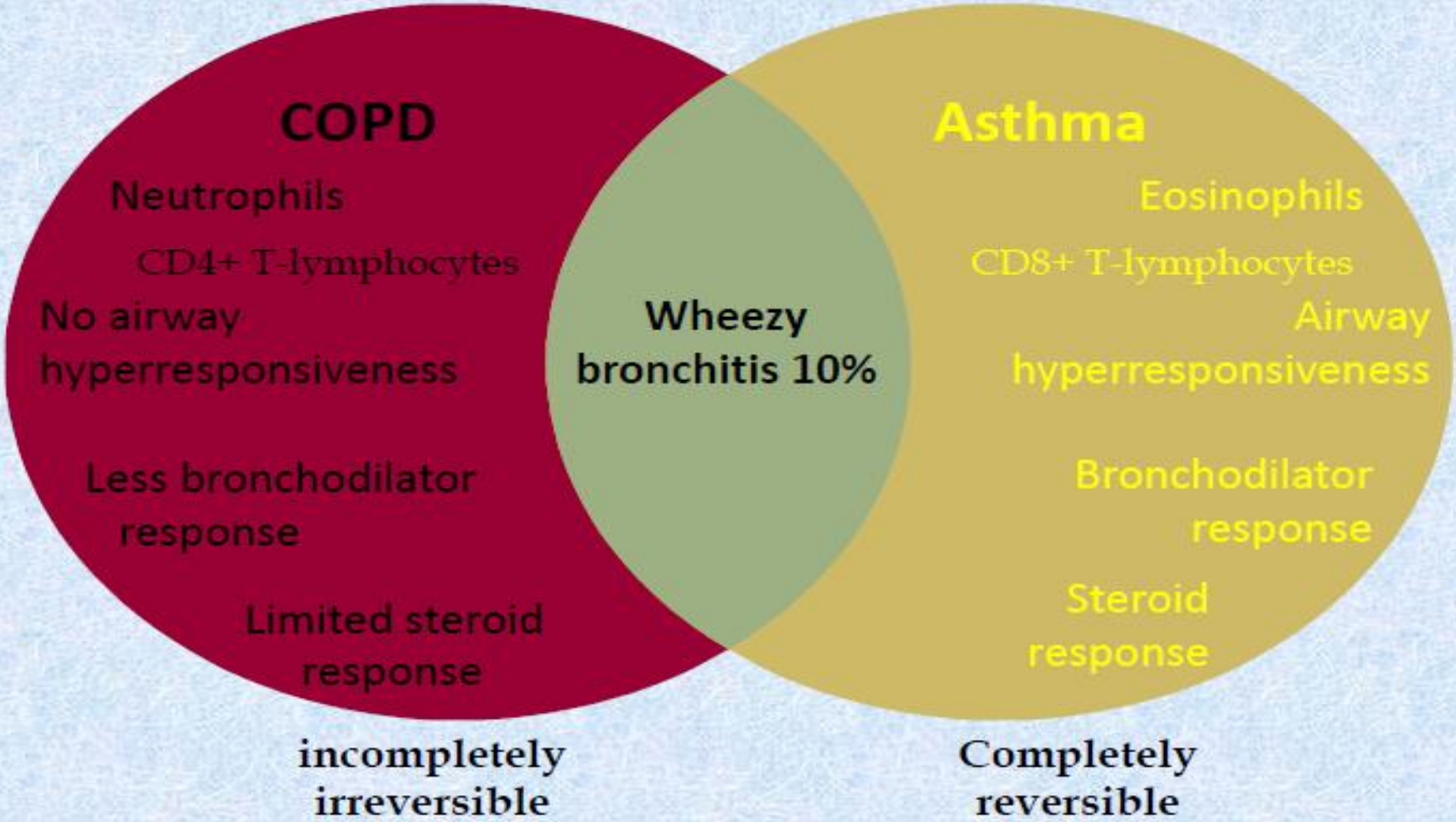
- Asthma
- Lung Cancer
- Tuberculosis
- Bronchiectasis
- Left Heart Failure
- Interstitial Lung Disease
- Cystic Fibrosis
- Idiopathic Cough

EXTRATHORACIC

- Chronic Allergic Rhinitis
- Post Nasal Drip Syndrome (PNDS)
- Upper Airway Cough Syndrome (UACS)
- Gastroesophageal Reflux
- n (e.g. ACE Inhibitors)

TABLE 2.2

Difference and overlap



Summary asthma versus COPD

	ASTHMA	COPD
age	early	Old age
Family hx	positive	Not common
Atopic hx	strong	Not common
Symptoms	Free in between attacks	Residual symptoms
wheezing	polyphonic	monophonic
course	Relapse, remittent	progressive
AHR	characteristic	no
spirometry	Reversible obstruction	Not reversible obstruction
KCO	Normal or increased	Normal or low
DLCO	normal	decrease
RV	Normal or increased	Usually increased

Diagnosis

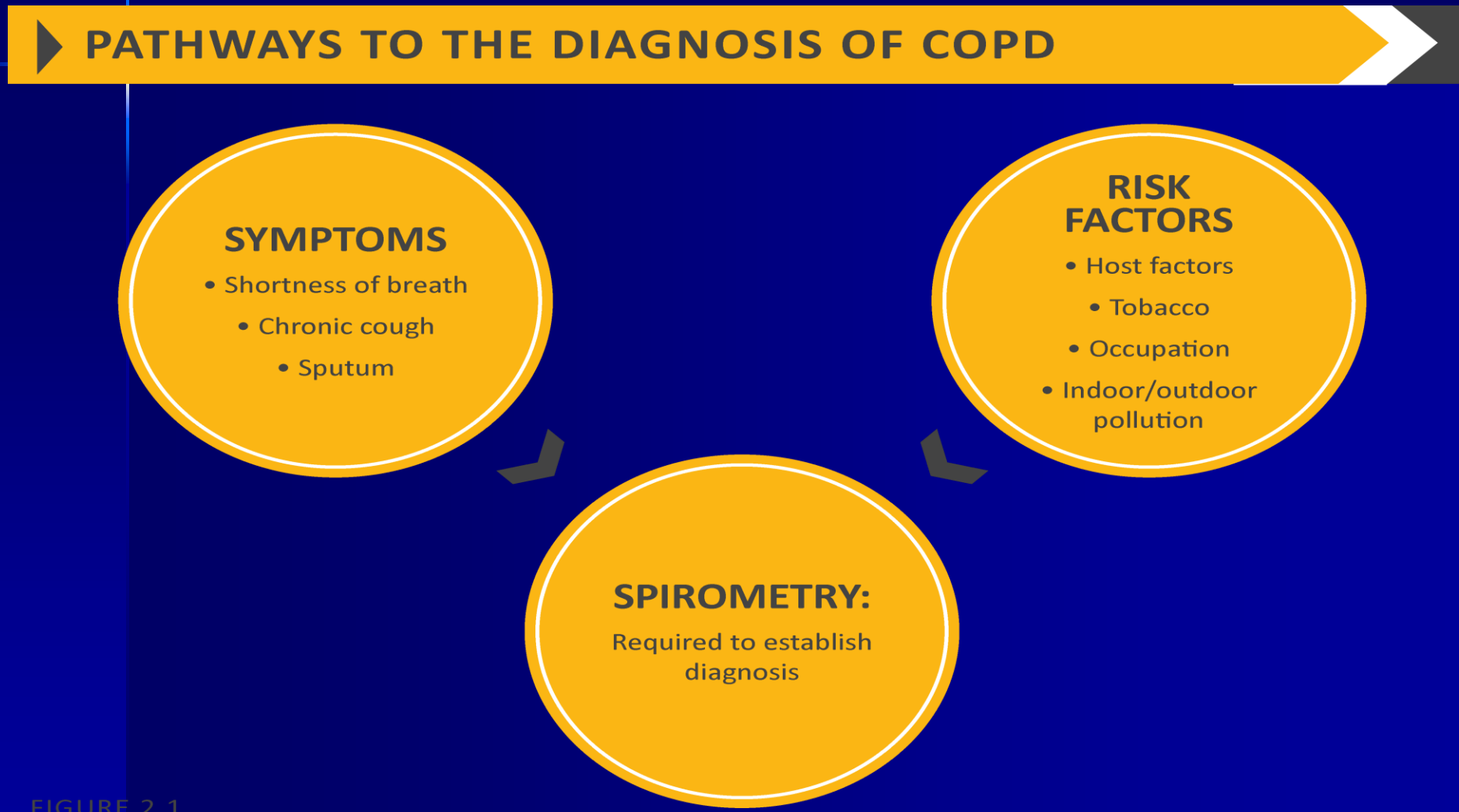


FIGURE 2.1

KEY INDICATORS FOR CONSIDERING A DIAGNOSIS OF COPD

Consider COPD, and perform spirometry, if any of these indicators are present in an individual over age 40. These indicators are not diagnostic themselves, but the presence of multiple key indicators increases the probability of a diagnosis of COPD. Spirometry is required to establish a diagnosis of COPD.

Dyspnea that is:	Progressive over time. Characteristically worse with exercise. Persistent.
Chronic Cough:	May be intermittent and may be unproductive. Recurrent wheeze.
Chronic Sputum Production:	Any pattern of chronic sputum production may indicate COPD.
Recurrent Lower Respiratory Tract Infections	
History of Risk Factors:	Host factors (such as genetic factors, congenital/developmental abnormalities etc.). Tobacco smoke (including popular local preparations). Smoke from home cooking and heating fuels. Occupational dusts, vapors, fumes, gases and other chemicals.
Family History of COPD and/or Childhood Factors:	For example low birthweight, childhood respiratory infections etc.

TABLE 2.1

Assessment of COPD patient

- Clinical
  - Degree of symptoms
  - Hx of exacerbations
  - Smoking
  - Comorbidity
- spirometry

Modified Medical Research Council (MRC) dyspnoea scale

Grade	Degree of breathlessness related to activities
0	No breathlessness, except with strenuous exercise
1	Breathlessness when hurrying on the level or walking up a slight hill
2	Walks slower than contemporaries on level ground because of breathlessness or has to stop for breath when walking at own pace
3	Stops for breath after walking about 100 m or after a few minutes on level ground
4	Too breathless to leave the house, or breathless when dressing or undressing

(MRC = Medical Research Council)

CAT™ ASSESSMENT

For each item below, place a mark (x) in the box that best describes you currently.
Be sure to only select one response for each question.

EXAMPLE: I am very happy	0 **X** 2 3 4 5	**I am very sad**	**SCORE**
I never cough	0 1 2 3 4 5	I cough all the time	
I have no phlegm (mucus) in my chest at all	0 1 2 3 4 5	My chest is completely full of phlegm (mucus)	
My chest does not feel tight at all	0 1 2 3 4 5	My chest feels very tight	
When I walk up a hill or one flight of stairs I am not breathless	0 1 2 3 4 5	When I walk up a hill or one flight of stairs I am very breathless	
I am not limited doing any activities at home	0 1 2 3 4 5	I am very limited doing activities at home	
I am confident leaving my home despite my lung condition	0 1 2 3 4 5	I am not at all confident leaving my home because of my lung condition	
I sleep soundly	0 1 2 3 4 5	I don't sleep soundly because of my lung condition	
I have lots of energy	0 1 2 3 4 5	I have no energy at all	

TOTAL SCORE:

Reference: Jones et al. ERJ 2009; 34 (3); 648-54.

FIGURE 2.3

Spirometric classification of COPD severity based on post-bronchodilator FEV1

PD FEV_1/ FVC	FEV_1% predicted	Severity of airflow obstruction post-bronchodilator: ATS/ERS (2004)	GOLD (2008)	NICE Clinical Guideline 101 (2010)
< 0.7	≥ 80%	Mild	Stage I — mild	Stage I — mild[1]
< 0.7	50–79%	Moderate	Stage II — moderate	Stage II — moderate
< 0.7	30–49%	Severe	Stage III — severe	Stage III — severe
< 0.7	< 30%	Very severe	Stage IV — very severe[2]	Stage IV — very severe[2]

[1]Mild COPD should not be diagnosed on lung function alone if the patient is asymptomatic. [2]Or FEV_1 < 50% with respiratory failure.
(ATS/ERS = American Thoracic Society/European Respiratory Journal; FEV_1 = forced expiratory volume in 1 sec; GOLD = Global Initiative for Chronic Obstructive Lung Disease; PD = post-bronchodilator)
Adapted from National Institute for Health and Care Excellence (NICE) CG101 — Chronic obstructive pulmonary disease in over 16s: diagnosis and management; 2010.

Calculation of the BODE index

Variable	Points on BODE index			
	0	1	2	3
FEV_1	≥65	50–64	36–49	≤35
Distance walked in 6 mins (m)	≥350	250–349	150–249	≤149
MRC dyspnoea scale*	0–1	2	3	4
Body mass index	>21	≤21		

A patient with a BODE score of 0–2 has a mortality rate of around 10% at 52 months, whereas a patient with a BODE score of 7–10 has a mortality rate of around 80% at 52 months.

THE REFINED ABCD ASSESSMENT TOOL

Spirometrically Confirmed Diagnosis

Post-bronchodilator $FEV_1/FVC < 0.7$

Assessment of airflow limitation

Grade	FEV_1 (% predicted)
GOLD 1	≥ 80
GOLD 2	50-79
GOLD 3	30-49
GOLD 4	< 30

Assessment of symptoms/risk of exacerbations

Moderate or Severe Exacerbation History

≥2 or ≥ 1 leading to hospital admission

0 or 1 (not leading to hospital admission)

C	D
A	B

mMRC 0-1 CAT < 10 | mMRC ≥ 2 CAT ≥ 10

Symptoms

GOLD 2023 ABE ASSESSMENT TOOL

- A and B
  - few exacerbation rate (1 or less moderate exacerbation not leading to hospitalization)
- A
  - Few symptoms (mMRC 0-1,CAT < 10)
- B
  - More symptoms (mMRC 2 or more ,CAT 10 or more
- E
  - Frequent exacerbation regardless of symptoms
  - 2 or more exacerbations or 1 or more severe

MANAGMENT OF STABLE COPD

Goals of treatment

- Symptoms reduce
  - Relieve symptoms
  - Improve exercise tolerance
  - Improved health status
- Risk reduce
  - Prevent disease progression
  - Prevent and treatment exacerbations
  - Reduce mortality

Components of treatment

- Pharmacological
- Non pharmacological
- Surgical

Pharmacological

- inhaled bronchodilators
  - SABA,SAMA
  - LABA,LAMA
- Inhaled steriods
- Oral bronchodilator therapy,such as theophylline preparations
- Orally selective phosphodiesterase inhibitors
- Oral steroids
- Vaccinations
- Vit D,azithromycin

INITIAL PHARMACOLOGICAL TREATMENT

≥ 2 moderate exacerbations or ≥ 1 leading to hospitalization	**Group C** LAMA	**Group D** LAMA or LAMA + LABA* or ICS + LABA** *Consider if highly symptomatic (e.g. CAT > 20) **Consider if eos ≥ 300
0 or 1 moderate exacerbations (not leading to hospital admission)	**Group A** A Bronchodilator	**Group B** A Long Acting Bronchodilator (LABA or LAMA)
	mMRC 0-1, CAT < 10	mMRC ≥ 2, CAT ≥ 10

FIGURE 4.2

GOLD 2023 new changes

- Group A
  - Bronchodilators (short acting or long acting)
  - Long acting is the preferred choice
- Group B
  - LAMA PLUS LABA
- Group E
  - LAMA PLUS LABA
  - Add ICS (LAMA PLUS LABA plus ICS) if blood eosinophil's more than 300 or indication for ICS

Inhaled steriods

- Should be considered in
  - ✓ History of 1 or more moderate exacerbations per year
  - ✓ History of of hospitalizations for exacerbations
  - ✓ Blood eosinophil's more than 100-300
  - ✓ Concomitant asthma
  - ❖ Should not used alone in COPD

Pharmacological

- Oral bronchodilator therapy,
- such as theophylline preparations, such as theophylline preparations, may be contemplated in patients who cannot use inhaled devices efficiently but their use may be limited by side-effects, unpredictable metabolism and drug interactions; hence the requirement to monitor plasma levels.

❖ selective phosphodiesterase inhibitors PDE4

➢ In svere and very severe copd with chronic bronchitis pattern with exacerbations (roflumilast)

❖ Oral glucocorticoids

➢ Should only in exacerbations and for short course

❖ Vaccinations

➢ annual influenza vaccination and, as appropriate, pneumococcal vaccination

❖ Vit D in deficient pt

❖ Azithromycin long term prescription

Nonpharmacological

- General

- Reducing exposure to noxious particles and gases
- Smoking cessation
  - remains the only strategy that impacts favourably on the natural history of COPD and improve survival .
- In regions where the indoor burning of biomass fuels is important, the introduction of non-smoking cooking devices or alternative fuels should be encouraged.

Nonpharmacological

- Oxygen therapy

- Long-term domiciliary oxygen therapy (LTOT) improves survival in selected patients with COPD complicated by severe hypoxaemia
- Indications Prescription of long-term oxygen therapy

Arterial blood gases are measured in clinically stable patients on optimal medical therapy on at least two occasions 3 weeks apart:

- PaO_2 <7.3 kPa (55 mmHg) irrespective of $PaCO_2$ and FEV_1 <1.5 L
- PaO_2 7.3–8 kPa (55–60 mmHg) plus pulmonary hypertension, peripheral oedema or nocturnal hypoxaemia
- the patient has stopped smoking

Use at least 15 hrs/day at 2–4 L/min to achieve a PaO_2 >8 kPa (60 mmHg) without unacceptable rise in $PaCO_2$

Nonpharmacological

- Pulmonary rehabilitation
- Exercise should be encouraged at all stages and patients reassured that breathlessness, while distressing, is not dangerous.
- Multidisciplinary programmes that incorporate physical training, disease education and nutritional counselling reduce symptoms,improve health status and enhance confidence.
- Most programmes include two to three sessions per week, last between 6 and 12 weeks, and are accompanied by demonstrable and sustained improvements in exercise tolerance and health status.

Surgical intervention

- Bullectomy
  - may be considered when large bullae compress

surrounding normal lung tissue.

- lung volume reduction surgery (LVRS)

Patients with predominantly upper lobe emphysema, preserved gas transfer and no evidence of pulmonary hypertension may benefit from LVRS which peripheral emphysematous lung tissue is resected with the aim of reducing hyperinflation and decreasing the work of breathing. Both bullectomy and LVRS can be performed thorascopically, minimising morbidity.

- Lung transplantation

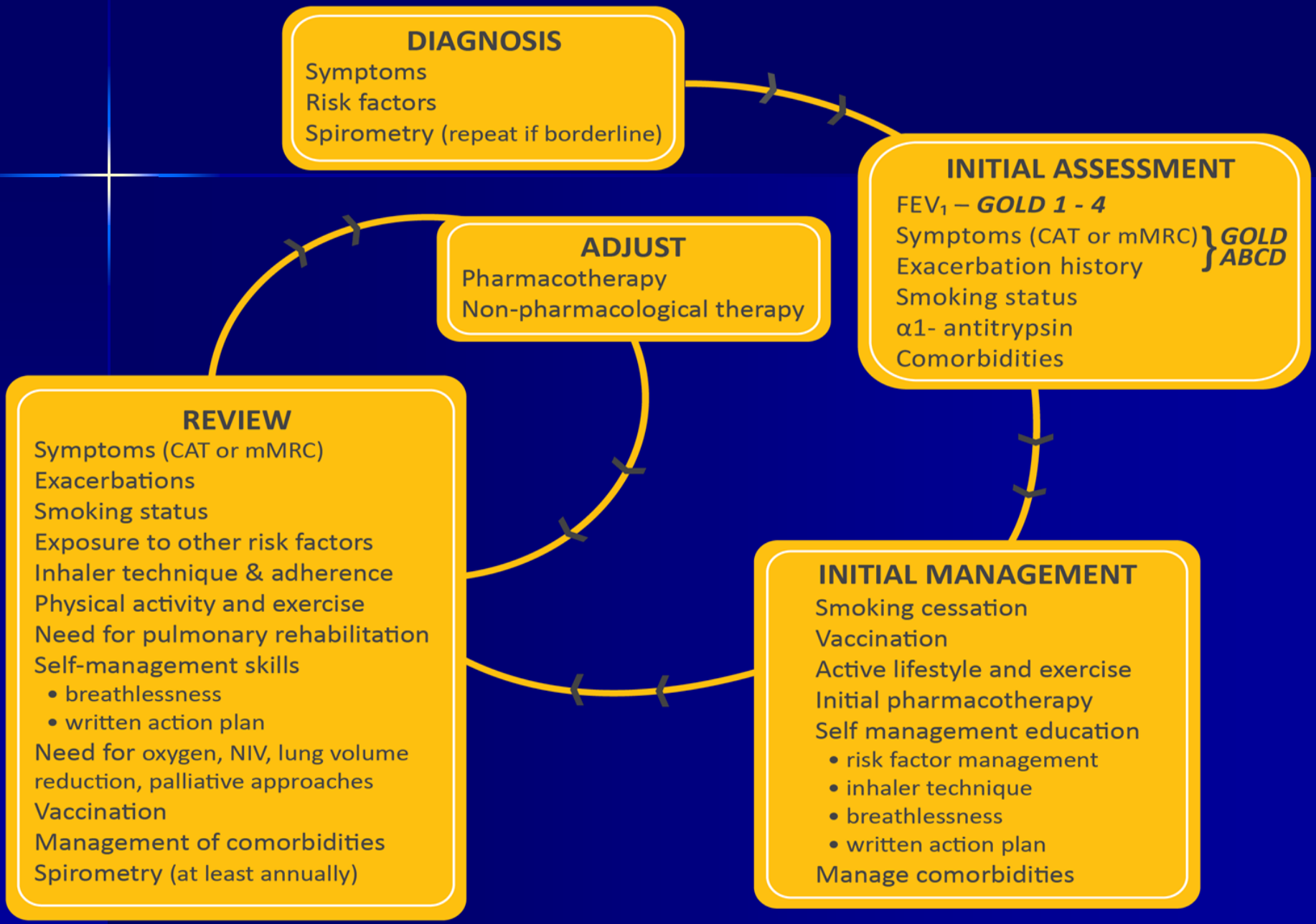
May benefit carefully selected patients with advanced disease

NON-PHARMACOLOGIC MANAGEMENT OF COPD*

PATIENT GROUP	ESSENTIAL	RECOMMENDED	DEPENDING ON LOCAL GUIDELINES
A	Smoking Cessation (can include pharmacologic treatment)	Physical Activity	Flu Vaccination Pneumococcal Vaccination
B, C and D	Smoking Cessation (can include pharmacologic treatment) Pulmonary Rehabilitation	Physical Activity	Flu Vaccination Pneumococcal Vaccination

*Can include pharmacologic treatment.

MANAGEMENT OF COPD



Acute exacerbations of COPD

Definition and triggers

- Definition

- Acute exacerbations of COPD are characterised by an increase in symptoms and deterioration in lung function and health status in < 14 days
- Exacerbations of COPD are important events in the management of COPD because they negatively impact health status, rates of hospitalization and readmission, and disease progression

- Triggers

- Exacerbations are mainly triggered by respiratory viral infections(most common is rhinovirus) although bacterial infections(H.Influena) and environmental factors such as pollution and ambient temperature may also initiate and/or amplify these events

Clinical presentation and investigations

- Increased dyspnea that is the key symptom of an exacerbation. Other symptoms include increased sputum purulence and volume, together with increased cough and wheeze.
- As other comorbidities that may worsen respiratory symptoms are common in COPD patients, clinical assessment to rule out differential diagnoses should be considered before diagnosis of a COPD exacerbation

DIFFERENTIAL DIAGNOSIS OF COPD EXACERBATION

WHEN THERE IS CLINICAL SUSPICION OF THE FOLLOWING ACUTE CONDITIONS, CONSIDER THE FOLLOWING INVESTIGATIONS:

PNEUMONIA

- Chest radiograph
- Assessment of C-reactive protein (CRP) and/or procalcitonin

PNEUMOTHORAX

- Chest radiograph or ultrasound

PLEURAL EFFUSION

- Chest radiograph or ultrasound

PULMONARY EMBOLISM

- D-dimer and/or Doppler sonogram of lower extremities
- Chest tomography – pulmonary embolism protocol

PULMONARY EDEMA DUE TO CARDIAC RELATED CONDITIONS

- Electrocardiogram and cardiac ultrasound
- Cardiac enzymes

CARDIAC ARRHYTHMIAS – ATRIAL FIBRILLATION/FLUTTER

- Electrocardiogram

Management

- Initial assessment
- Hx
- Examination (to exclude alternative dx),severity
- Check oxygen saturation and ABG if needed
- Assess severity
- Assess the need for hospitalization
- Give oxygen and bronchodilators

POTENTIAL INDICATIONS FOR HOSPITALIZATION ASSESSMENT*

- Severe symptoms such as sudden worsening of resting dyspnea, high respiratory rate, decreased oxygen saturation, confusion, drowsiness.
- Acute respiratory failure.
- Onset of new physical signs (e.g., cyanosis, peripheral edema).
- Failure of an exacerbation to respond to initial medical management.
- Presence of serious comorbidities (e.g., heart failure, newly occurring arrhythmias, etc.).
- Insufficient home support.

*Local resources need to be considered.

Pharmacological treatment

- Bronchodilators
- it is recommended that short-acting inhaled beta2-agonists, with or without short-acting anticholinergics, are the initial bronchodilators for acute treatment of a COPD exacerbation.
- use the MDI inhaler one or two puffs every one hour for two or three doses and then every 2-4 hours,or neublizer based on the patient's response.
- Glucocorticoids
- A dose of 40 mg prednisone per day or iv hydrocortison or methyl prednisolon for 5 days is recommended.

Pharmacological treatment

- Antibiotics

- should be given to patients with exacerbations of COPD who have three cardinal symptoms:increase in dyspnea, sputum volume, and sputum purulence; have two of the cardinal symptoms, if increased purulence of sputum is one of the two symptoms; require mechanical ventilation (invasive or noninvasive).
- The recommended length of antibiotic therapy is 5-7 days
- Usually initial empirical treatment is an aminopenicillin with clavulanic acid, macrolide, or tetracycline.
- In patients with frequent exacerbations, severe airflow limitation, and/or exacerbations requiring mechanical ventilation, cultures from sputum or other materials from the lung should be performed.

Pharmacological treatment

- Adjunct therapies
  - an appropriate fluid balance,
  - use of diuretics when clinically indicated
  - anticoagulants,
  - treatment of comorbidities and nutritional aspects

Respiratory support

- Oxygen therapy.
  - This is a key component of hospital treatment of an exacerbation. Supplemental oxygen should be titrated to improve the patient's hypoxemia with a target saturation of 88-92%.
  - Venturi masks (high-flow devices) offer more accurate and controlled delivery of oxygen than do nasal prongs.

Respiratory support

INDICATIONS FOR RESPIRATORY OR MEDICAL INTENSIVE CARE UNIT ADMISSION*

- Severe dyspnea that responds inadequately to initial emergency therapy.
- Changes in mental status (confusion, lethargy, coma).
- Persistent or worsening hypoxemia ($PaO_2 < 5.3$ kPa or 40mmHg) and/or severe/worsening respiratory acidosis (pH < 7.25) despite supplemental oxygen and noninvasive ventilation.
- Need for invasive mechanical ventilation.
- Hemodynamic instability - need for vasopressors.

*Local resources need to be considered.

INDICATIONS FOR NONINVASIVE MECHANICAL VENTILATION (NIV)

At least one of the following:

- Respiratory acidosis ($PaCO_2 \geq 6.0$ kPa or 45 mmHg and arterial pH ≤ 7.35).
- Severe dyspnea with clinical signs suggestive of respiratory muscle fatigue, increased work of breathing, or both, such as use of respiratory accessory muscles, paradoxical motion of the abdomen, or retraction of the intercostal spaces.
- Persistent hypoxemia despite supplemental oxygen therapy.

TABLE 5.6

INDICATIONS FOR INVASIVE MECHANICAL VENTILATION

- Unable to tolerate NIV or NIV failure.
- Status post - respiratory or cardiac arrest.
- Diminished consciousness, psychomotor agitation inadequately controlled by sedation.
- Massive aspiration or persistent vomiting.
- Persistent inability to remove respiratory secretions.
- Severe hemodynamic instability without response to fluids and vasoactive drugs.
- Severe ventricular or supraventricular arrhythmias.
- Life-threatening hypoxemia in patients unable to tolerate NIV.

DISCHARGE CRITERIA AND RECOMMENDATIONS FOR FOLLOW-UP

- Full review of all clinical and laboratory data.
- Check maintenance therapy and understanding.
- Reassess inhaler technique.
- Ensure understanding of withdrawal of acute medications (steroids and/or antibiotics).
- Assess need for continuing any oxygen therapy.
- Provide management plan for comorbidities and follow-up.
- Ensure follow-up arrangements: early follow-up < 4weeks, and late follow-up < 12weeks as indicated.
- All clinical or investigational abnormalities have been identified.

Follow up

1 – 4 WEEKS FOLLOW-UP

- Evaluate ability to cope in his/her usual environment.
- Review and understanding treatment regimen.
- Reassessment of inhaler techniques.
- Reassess need for long-term oxygen.
- Document the capacity to do physical activity and activities of daily living.
- Document symptoms: CAT or mMRC.
- Determine status of comorbidities.

12 – 16 WEEKS FOLLOW-UP

- Evaluate ability to cope in his/her usual environment.
- Review understanding treatment regimen.
- Reassessment of inhaler techniques.
- Reassess need for long-term oxygen.
- Document the capacity to do physical activity and activities of daily living.
- Measure spirometry: FEV_1.
- Document symptoms: CAT or mMRC.
- Determine status of comorbidities.

Remember

- How to diagnose COPD
- Asthma versus COPD
- Haemptysis,loss of weight ,clubbing are not common and should look for other cause
- Choice of treatment according to stage
- Inhaled steriods should not be used alone in COPD
- How to deal with exacerbations

Suggested readings and home activity

- Review pt plan of treatment
- Indications for alph antitrypsine D screening
- COPD versus HF

Thank you